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MICROSCOPIC FEATURES OF LEUKEMIC INFILTRATION IN PERIPHERAL ORGANS (LIVER, SPLEEN, LYMPH NODES)

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Annotation: Leukemia is a malignant neoplasm of hematopoietic origin characterized by the uncontrolled proliferation of abnormal leukocytes within the bone marrow and their subsequent spread to peripheral organs. The microscopic features of leukemic infiltration in organs such as the liver, spleen, and lymph nodes provide valuable diagnostic and prognostic information regarding disease stage and aggressiveness. Leukemic cells invade these tissues through hematogenous dissemination, replacing normal parenchyma and altering organ architecture. Histologically, infiltration patterns vary between acute and chronic, myeloid and lymphoid forms of leukemia, ranging from diffuse and destructive replacement to nodular or sinusoidal involvement. Recognition of these morphological patterns under light microscopy is essential for differential diagnosis, disease monitoring, and understanding leukemic pathophysiology.

Key words: leukemia, histopathology, liver, spleen, lymph nodes, infiltration, blasts, hematologic malignancy

Introduction

Leukemia primarily originates in the bone marrow but often extends to peripheral organs as the disease progresses. This extramedullary spread reflects the systemic nature of hematologic malignancies. The liver, spleen, and lymph nodes, as highly vascular and immune-active organs, are common targets for leukemic infiltration. Their microscopic examination not only confirms leukemic dissemination but also helps to differentiate leukemia from other hematolymphoid disorders such as lymphoma or myelodysplasia. The histopathological features of infiltration depend on the type and chronicity of leukemia, the degree of blast proliferation, and the host tissue's microenvironmental response.

Microscopic Features in the Liver

The liver is one of the most frequently involved organs in leukemia due to its rich sinusoidal blood supply and reticuloendothelial function. Under normal conditions, the hepatic lobule consists of radiating cords of hepatocytes separated by sinusoids lined by endothelial and Kupffer cells. In leukemia, these structures become distorted as malignant cells infiltrate through sinusoidal and portal pathways.

In acute leukemias, infiltration is typically diffuse and sinusoidal. Leukemic blasts accumulate within hepatic sinusoids, expanding and compressing adjacent hepatocytes. The infiltrating cells are large, round, with high nuclear-to-cytoplasmic ratios, fine chromatin, and prominent nucleoli. Portal tracts are often expanded by clusters of blasts, and the lobular pattern may become effaced. Kupffer cell hyperplasia and mild hepatocellular atrophy are common secondary findings. In some cases, small foci of leukemic necrosis may appear due to vascular obstruction.

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In chronic leukemias, particularly chronic lymphocytic leukemia (CLL), the pattern of infiltration is nodular or periportal. Small, mature lymphocytes with dense chromatin accumulate in the portal areas without significant destruction of hepatocytes. In late stages, these nodules may coalesce, leading to diffuse portal fibrosis. The hepatic cords usually remain intact, and the infiltration tends to be non-destructive. Special stains such as reticulin may reveal increased perisinusoidal fibers, while immunohistochemistry confirms the lineage of infiltrating cells through markers such as CD20 (B-cells) or MPO (myeloid cells).

Microscopic Features in the Spleen

The spleen is another organ frequently affected by leukemic infiltration due to its filtration and immune surveillance functions. Histologically, the normal spleen consists of white pulp (lymphoid nodules and periarteriolar sheaths) and red pulp (splenic cords and sinusoids). Leukemia disrupts this architecture through various patterns of infiltration.

In acute leukemias, the spleen shows diffuse involvement of red pulp with massive accumulation of blasts that obliterate the sinusoidal network. The white pulp may appear atrophic due to compression by infiltrating cells. The splenic cords are filled with immature hematopoietic cells, leading to congestion and sometimes infarction. The capsule and trabeculae may be thickened due to reactive fibrosis.

In chronic leukemias, particularly CLL and chronic myeloid leukemia (CML), the infiltration pattern is more organized and nodular. In CLL, small lymphocytes infiltrate the white pulp and replace the normal follicles with monotonous cellular aggregates. In contrast, CML shows marked extramedullary hematopoiesis, with proliferation of granulocytic precursors, megakaryocytes, and occasional erythroid elements within the red pulp. This myeloid metaplasia reflects the marrow's compensatory attempt to maintain hematopoiesis.

Microscopically, megakaryocytes appear as large cells with multilobed nuclei scattered throughout the red pulp. The white pulp may show hyperplasia in early disease but eventually becomes effaced as the process progresses.

Microscopic Features in the Lymph Nodes

Lymph node involvement in leukemia demonstrates variable histological patterns depending on the type of leukemia. Normal lymph node architecture consists of a cortex (with lymphoid follicles), a paracortex, and a medulla with cords and sinuses.

In acute lymphoblastic leukemia (ALL), the lymph nodes show diffuse effacement of normal architecture by monotonous sheets of lymphoblasts. These cells are small to medium-sized with high nuclear-to-cytoplasmic ratios, fine chromatin, and inconspicuous nucleoli. Mitotic figures are frequent, reflecting high proliferative activity. The sinuses are obliterated, and residual follicles are absent.

In acute myeloid leukemia (AML), infiltration occurs as diffuse sheets of myeloblasts with abundant cytoplasm, fine azurophilic granules, and occasional Auer rods. The lymph node architecture is similarly destroyed, but myeloid infiltrates can sometimes be identified by their positive reaction to myeloperoxidase (MPO) and lysozyme stains.

In chronic lymphocytic leukemia (CLL), the lymph nodes show a pseudofollicular pattern composed of small mature lymphocytes with condensed chromatin. Pale-staining proliferation

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centers (pseudofollicles) are often present, containing prolymphocytes and paraimmunoblasts. Unlike lymphoma, these nodes usually retain partial sinus structure.

In chronic myeloid leukemia (CML), nodal infiltration is less common but may present with extramedullary myelopoiesis, including the presence of myelocytes and megakaryocytes.

Comparative Observations

Across these organs, certain microscopic principles remain consistent. Acute leukemias exhibit diffuse, destructive, and poorly differentiated infiltration with high blast counts and loss of tissue organization. Chronic leukemias, on the other hand, demonstrate more gradual and often nodular infiltration by mature or maturing cells, preserving some aspects of normal architecture. Sinusoidal infiltration and fibrosis are common reactive changes, and extramedullary hematopoiesis is particularly prominent in chronic myeloid leukemia.

The identification of leukemic infiltration requires careful histological examination supplemented by cytochemical and immunohistochemical stains. Markers such as CD3, CD20, CD34, MPO, and TdT aid in lineage determination and differentiation from lymphoma or reactive hyperplasia.

Conclusion

Leukemic infiltration of peripheral organs reflects the systemic progression of hematopoietic malignancy and exhibits distinct microscopic patterns according to leukemia type and chronicity. In the **liver**, infiltration is primarily sinusoidal and periportal, leading to distortion of lobular structure and vascular congestion. In the **spleen**, the red pulp becomes expanded with leukemic cells, while white pulp may undergo atrophy or myeloid metaplasia. In the **lymph nodes**, normal architecture is replaced by monotonous populations of blasts or mature lymphocytes, depending on lineage and stage.

These histopathological changes not only confirm extramedullary dissemination but also help distinguish between acute and chronic, myeloid and lymphoid forms of leukemia. Recognition of infiltration patterns under microscopy remains critical for diagnosis, staging, and evaluation of therapeutic response. The integration of histological features with immunohistochemistry and molecular data allows a comprehensive understanding of leukemic organ involvement and its clinical implications.

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